The management of traumatic brain injury

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Abstract

Traumatic brain injury (TBI) remains a significant global problem with an increasing socioeconomic impact. Increasing knowledge of the pathophysiology of TBI has led to a systematic multidisciplinary approach towards management aiming to protect the brain from secondary injury. Early management starting from the scene of injury, to intensive care and surgical settings is paramount to achieve this purpose. TBI includes a large spectrum of diseases, therefore identifying the correct pathology on imaging is fundamental to define the appropriate next steps of management. Computed tomography (CT) imaging to date remains the gold standard for initial radiological assessment. Intracranial and cerebral perfusion pressure targeted therapies are still the minimum requirement in most of modern intensive care units worldwide. Decompressive craniectomy is a fundamental technique to control medically refractory intracranial hypertension and reduce mortality; however, its burden in terms of outcomes remains a controversial topic requiring further debate. There is emerging evidence that TBI is a chronic illness, with increased incidence of cognitive and behavioural deficits, neurodegenerative disease such as seizures and epilepsy, and an increased mortality that extends well beyond the initial TBI stage. Ongoing research into novel biomarkers, the application of artificial intelligence (AI) and an increasing global effort may yield future therapeutic strategies to improve clinical outcomes.

Keywords Decompressive craniectomy; intracranial haematoma; intracranial pressure; management; neurocritical care; neuroradiology; operative management; traumatic brain injury

Introduction

Traumatic brain injury (TBI) is becoming increasingly recognized as being responsible for a substantial proportion of the global burden of disease, especially in low- to middle-income countries (LMICs). The impact of TBI is extremely large, and is currently estimated to affect between 27 and 69 million people per year. Many authors also acknowledge that the incidence of TBI is underreported.[1–3] TBI is the most significant cause of neurodisability currently and in the foreseeable future, and its global incidence is rising despite improving efforts to understand strategies for injury prevention.[4–6] The pattern of TBI seems to be constantly evolving, such as seen with increasing motor vehicle use in low- and middle-income countries, and with falls amongst an increasingly older population in high-income countries.[4,5] Road traffic accidents and falls remain the most common causative mechanism for TBI in high-income countries accounting for more than 50% of injuries, and there is an increasing incidence of sports-related TBI, particularly in young people.[1] TBI is more common in men than women across all age groups.[1]

The pathophysiology of TBI has been described in relation to the Monro–Kellie doctrine since the 18th century. However, with increasing sophistication of neuroimaging and understanding of neurochemical cascades, a purely mechanical understanding of TBI pathophysiology is now insufficient. Nevertheless, a biomechanical background remains important to adopt effective treatment strategies aimed at minimizing the damage suffered from the primary TBI and subsequent secondary brain injury.[7] With TBI, force is transmitted to the cranial cavity in a variety of ways. Force can be transmitted in a rotational manner, in an acceleration–deceleration fashion, or both. Damage and force transmission can also occur with penetration, such as in missile injury. As force is transmitted into the cranial cavity there is stretch and strain of neural and vascular structures, causing direct injury to brain tissue (axonal stretch injury) or blood vessels (causing extravasation of blood, resulting in intracranial haemorrhage and a pathological increase in intracranial blood volume).[8]

The Monro–Kellie doctrine of intracranial pressure (ICP) states that for any increase in volume of one of the skull's contents, being brain, blood and cerebrospinal fluid (CSF), there must be a compensatory decrease in volume of one or both of the other two contents in order to maintain a constant ICP. As compensatory mechanisms become exhausted and as volume of one component increases, ICP begins to rise exponentially within the fixed volume of the closed skull (Figure 1a). The consequences of raised ICP include a reduction in the mean pressure of arterial blood perfusing the brain, herniation of the brain between intracranial compartments, and altered cerebral compliance. Cerebral perfusion pressure (CPP) is a surrogate measure of cerebral blood flow and is calculated by deducting ICP from mean arterial blood pressure. As ICP rises CPP can be maintained by autoregulatory mechanisms in compensation, but as autoregulation becomes exhausted CPP will fall as ICP rises, putting the patient at risk of secondary hypoxic brain injury (Figure 1b).

Herniation syndromes are well recognized and can also lead to secondary brain injury from arterial compression and territorial ischaemia or from direct brainstem compression. An increase in volume of any of the skull contents can occur following TBI; however, most commonly it is an increase in the amount of blood in the form of haematoma, or the amount of brain in the form of cerebral oedema, or frequently both. Correction of pathological volumes in TBI can allow manipulation of ICP and

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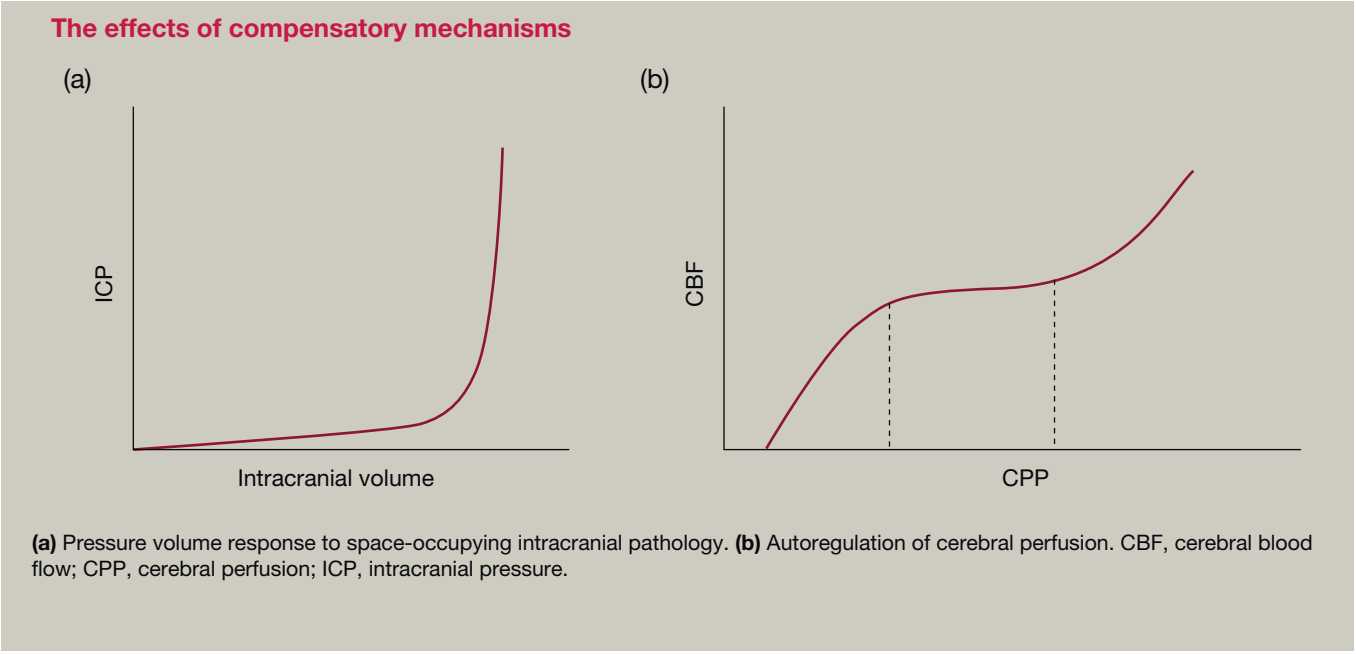

(a) Pressure volume response to space-occupying intracranial pathology. **(b)** Autoregulation of cerebral perfusion. CBF, cerebral blood flow; CPP, cerebral perfusion; ICP, intracranial pressure.

Figure 1

CPP and can prevent herniation syndromes with minimization of secondary brain injury.[9]

Less well understood are the cellular and molecular pathways associated with neuroinflammation in TBI. These pathways may involve activation of microglia, increased extracellular levels of excitatory amino acids such as glutamate, accumulation of abnormal protein plaque aggregates including b-amyloid precursor protein (APP) or phosphorylated-tau (P-tau), activation of reactive oxygen and nitrogen species, and other less understood neuroinflammatory cascades.[1] There may also be genetic factors that influence the cellular response to TBI,[1,5,10] which may help explain the different individual clinical outcomes across a cohort of patients with seemingly similar pathology and cerebral insults. Further studies into molecular pathogenesis and cerebral microdialysis following TBI may yield a better understanding of these pathways and the production of novel therapeutic agents to minimize brain injury at a cellular level.

Resuscitation and initial management

Early management of TBI has a key role in a patient's outcome and it should be initiated from the scene with a systematic approach. The traumatized brain is highly susceptible to secondary brain injury from hypoxia, hypothermia and hypotension, and for the vast majority of TBI sufferers it is secondary injury that management is targeted towards preventing. Hypotension reduces cerebral perfusion pressure to the injured brain and has a profound negative effect on outcome. Even brief periods of hypoxia and hypotension are harmful to the injured brain, and when they occur together they create a larger negative effect on outcome than either in isolation. Prehospital care of the TBI patient aims to optimize brain perfusion while rapidly transporting the patient to a location where he or she can receive definitive care. Current evidence suggests that the historical treatment thresholds for oxygen saturation and/or blood pressure are likely too low.[11] Stronger emphasis on avoiding the threshold 'region' rather than focusing exclusively on waiting to treat already established low values is most likely appropriate in the absence of more conclusive evidence, but is yet to be demonstrated in a randomized study. To minimize secondary brain injury, prehospital healthcare professionals should continuously monitor for, anticipate, prevent, and rapidly correct both hypoxia and hypotension in patients with suspected TBI.[12]

Clinical classification of TBI severity still relies on the now 50-year-old Glasgow Coma Scale (GCS) score (Table 1) which remains a simple but very effective tool in clinical management.

The Glasgow Coma Scale score. Numbers for best response to the three individual behaviour categories are added to derive a total score out of 15

Behaviour	Response	Score
Eye opening	Spontaneously	4
	To speech	3
	To pain	2
	No response	1
Best verbal	Oriented to time, place and person	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No response	1
Best motor	Obeys commands	6
	Localizes to pain	5
	Flexion withdrawal from pain	4
	Abnormal flexion (decorticate)	3
	Abnormal extension (decerebrate)	2
	No response	1

Table 1

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GCS consists of three components: best eye-opening response, best verbal response, and best motor response. Adding the three components of GCS provides a score ranging from 3 to 15, although when documenting GCS it is more informative to demonstrate the individual components, such as E2V4M5 = 11/15. Of the three GCS components, the motor response has the highest prognostic information when compared to the complete score. Authors have recognized limitations in accuracy and inter-observer reliability in performing GCS assessments, and particularly with prehospital scores. Most commonly, TBI is thought of as being mild (GCS 13/15), moderate (GCS 9/12) or severe (GCS $\leq$8), and by definition GCS of 8 or less is equivalent to coma. Although a useful, widespread and reproducible scoring system, it is ultimately not sophisticated enough to convey much more information than the patient's level of alertness following TBI, and provides no understanding of the underlying pathology. There is likely some prognostic information between initial post-resuscitation GCS and clinical outcome; however, other factors such as raised ICP and radiological pattern of brain injury are more closely linked to long-term outcome. In addition to classifying injury severity, documentation of the initial GCS as well as repeated reassessment of the conscious state is useful to demonstrate clinical improvement or decline. A person suffering a concussion for instance may have an initial GCS of 3 that steadily improves to 15, whereas someone with a rapidly expanding extradural haematoma could demonstrate the reverse GCS trend.[12] A transitory impairment of conscious state may imply the presence of post-traumatic seizures (PTS), this event should always be accounted in the initial management of TBI, since it may be misleading for the following management.[13] Prior to making a radiological diagnosis of TBI the classification of TBI severity is useful in planning initial management and also in triaging patient priorities.

Together with airway protection, breathing and circulation resuscitation, the patient's vital signs should be closely monitored to detect and prevent systemic shock. Bradycardia with hypertension may be a sign of raised ICP; this is the Cushing response, first recognized by pioneering American neurosurgeon Harvey Cushing, and is a physiological increase in cardiac stroke volume in an attempt to maintain cerebral perfusion following an abnormal increase in ICP. Another important physical feature to make note of is pupil size and reaction to light. Pupil size, symmetry, and reactivity are affected by many different neuro-anatomical pathways and are supported by the literature as integral to clinical decision making, acute management, and long-term prognosis of TBI. A single fixed and dilated pupil may reflect oculomotor nerve compression at the tentorium secondary to an ipsilateral space occupying hemispheric pathology, prompting rapid resuscitation and surgical intervention, whereas bilaterally fixed pupils are generally thought to be associated with more widespread brainstem injury and a poorer prognosis. There can be extracerebral explanations for pupil inequality; anisocoria can occur in TBI secondary to globe injury and traumatic mydriasis or carotid injury and acquired Horner's syndrome, and sometimes a fixed pupil ultimately proves to be a glass eye. Pupil size and reaction do not make up part of the GCS assessment and must be documented separately.

Care must be taken with the cervical spine until a fracture or unstable spine injury has been excluded, and a cervical injury should always be suspected even with seemingly relatively minor cranial trauma. A spinal cord injury can also be a contributing factor in shock or could confound the motor component of the coma examination. Inline spinal immobilization in a hard cervical collar and spinal precautions should be taken during the initial management stage, and this can occasionally prove to be difficult in cases of extrication from a motor vehicle for instance. Collars must not be too firmly fitted as this may occlude venous outflow or compress the cervical carotid arteries. A brief survey of the head and neck may provide clues to the underlying pathology, such as bruising around the eyes (raccoon or panda eyes) or posterior-auricular region (Battle's sign) which may indicate a skull base fracture, scalp lacerations or degloving injuries with bone or brain tissue on view, or blood and/or CSF from the nose or ears.

Injury recognition and radiology

TBI is an umbrella term including one or more different conditions in isolation or combination. Clinical history and medical examination may sometimes guide the clinician towards the identification of one particular intracranial pathology following TBI, however imaging is what determines the accurate diagnosis and will direct the following management. The gold standard for radiological recognition of TBI is computed tomography (CT) scan. CT will accurately demonstrate the presence and location of acute blood products, skull fractures, vascular injury, intracranial gas, foreign bodies, and extracranial soft tissue injuries. Other suspected extracranial injuries can also be investigated for during the same examination, such as spine fractures. Accessibility and rapidity still make CT the imaging choice in the majority of trauma centres (Figure 2). MRI can be useful in later management and particularly with injury prognostication, but it should not be considered of primary importance in the acute setting.[14]

CT scan patterns identified include skull fractures, hematomas (both intra- and extra-axial), and diffuse brain injury. In many TBI patients an initial CT scan may show no apparent abnormality. Skull fractures can be classified as involving the vault or the skull base, non- or minimally displaced or depressed, and/or open or closed. Gas may be visualized within the cranial cavity, which raises the possibility of a dural breach. Fractures along the skull base foramina can encroach upon vascular structures, and CT angiography is helpful to determine if there is arterial compromise or injury. Similarly, skull vault or posterior fossa fractures can be associated with dural venous sinus tear or occlusion, and a CT venogram can be utilized to investigate for sinus injury. Vault fractures, particularly over the squamous temporal bone, may be associated with extra-axial haematomas, the classic example being an extradural haematoma from laceration of a branch of the middle meningeal artery at the pterion.[15]

Intracranial haematomas are either extra-axial (outside of the brain) or intra-axial (within the brain substance, also known as intracerebral or intraparenchymal). Extra-axial haematomas are broadly classified to their relation to the meninges as being either an extradural haematoma (EDH), subdural haematoma (SDH) or subarachnoid haematoma (SAH). The three patterns may co-exist, sometimes in multiple areas of the brain. The source of blood from EDH is generally arising from an injured dural artery or epidural veins, and although the dura is quite firmly adherent

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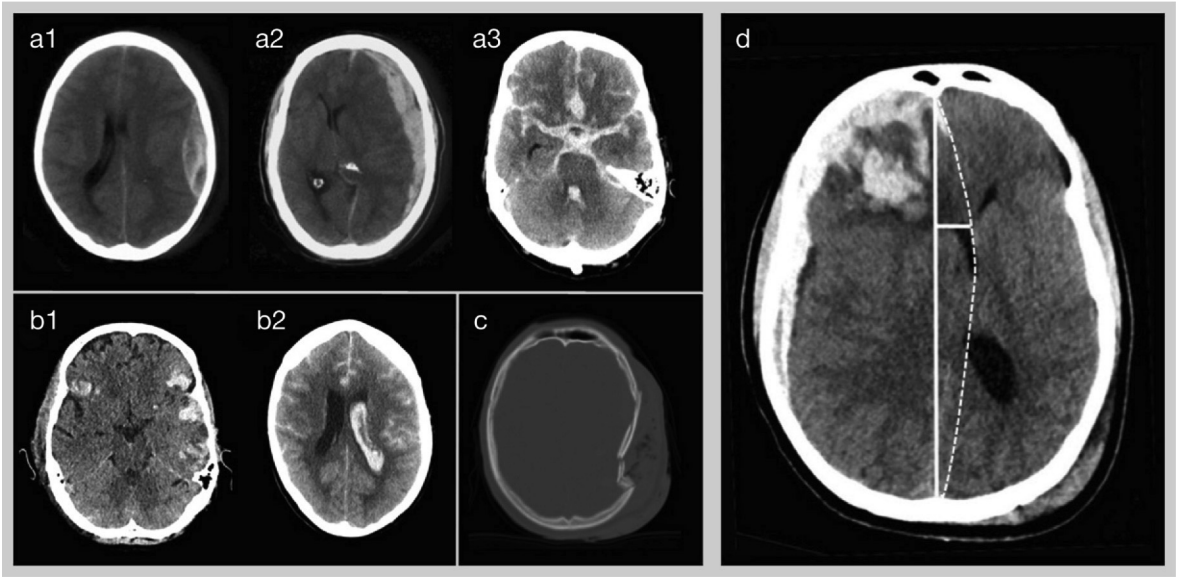

Figure 2 CT scan classification of TBI. **a**, Extra-axial injuries (**a**1, EDH; **a**2, SDH; **a**3, SAH). **b**, Intra-axial injuries (**b**1, ICH; **b**2, IVH). **c**, Skull vault fracture with depression. **d**, Measuring midline shift at the level of the interventricular foramen; the anatomical midline is represented by the vertical solid line, the pathological midline by the dashed line, and the amount of midline shift by the horizontal solid line. CT, computerized tomography; EDH, extradural haematoma; ICH, intracerebral haematoma; IVH, intraventricular haemorrhage; SAH, subarachnoid haemorrhage; SDH, acute subdural haematoma.

to the inner skull, continued haematoma expansion from high arterial blood pressure strips the dura from the bone, producing a classical biconvex haematoma appearance. The underlying brain is compressed from the increasing volume of the haematoma. However, the brain is separated from the blood by the dura, and often the brain itself is macroscopically normal.[16]

In contrast, the source of bleeding in SDH is generally from brain surface bridging veins or from contused brain itself. Bleeding generally occurs at a slower pace than EDH; however, it may be often associated with cerebral oedema from the injured brain. The blood products are located beneath the dura and blood collects over the cerebral convexity, with haematoma expansion unrestricted by dural adherence to skull as in EDH. There is mass effect not only from the volume of the SDH, but also from the adjacent swollen brain. SDH may also be associated with traumatic sub-arachnoid haemorrhage (tSAH). tSAH is the most common cause of subarachnoid haemorrhage, and generally arises from tearing of small caliber vessels bridging within the subarachnoid space. It must be distinguished from aneurysmal subarachnoid haemorrhage which may be radiologically identical, and confusingly may also be associated with a collapse and head strike. Unlike aneurysmal SAH, traumatic SAH is rarely associated with delayed ischaemic neurological deficits, however in situations in which the precipitating event is unclear then further vascular imaging such as CT angiogram may be appropriate to investigate for an underlying vascular pathology.

Intra-axial blood is recognized as being parenchymal (contusion) or intraventricular (IVH). A contusion may be focal with distinct margins as with other types of non-traumatic intra-cerebral haemorrhage, or may present as diffuse multifocal clusters of contusions. Buoyant within the cerebrospinal fluid (CSF) the brain is susceptible to motion following TBI, such that with transmission of force to the head the brain will have a degree of inertia to move within the skull, producing an internal impact between brain and the inner skull table.[17] This explains the gliding pattern of contusion commonly seen along the inferior frontal lobes and anterior temporal lobes referred to as an indirect or 'contre-coup' injury, following a posterior head strike (the direct or 'coup' injury). IVH may be associated with hydrocephalus in the event that blood products are limiting free movement of CSF between the ventricular compartments, either by mechanically occluding one of the foramens or blocking the transependymal resorption of CSF.

In addition to identifying fractures, gas, blood products and foreign objects, the CT scan is useful to determine brain swelling, which may be focal or generalized. The Marshall classification of TBI quantifies diffuse brain injury in TBI by assessing occlusion of basal CSF cisterns, midline shift, and high or mixed density lesions of 25 ml, and was designed to aid in the prediction of outcome following TBI. Other CT-based classification systems have been introduced in order to improve the prognostic value of CT in TBI, such as the Rotterdam score; however, neither system has been shown to perform as well as multivariable prognostication models,[18] and may be best suited to guiding clinical decision making.

Surgical management

Once an injury pattern has been recognized, it is important to quickly determine if surgical intervention is warranted, which is done in conjunction with the clinical state of the patient. Indications for surgical intervention have been published by the Brain Trauma Foundation (BTF), and these guidelines provide clear recommendations to aid surgical decision making based on objective CT scan data and clinical findings.[19] A common operation in TBI to manage intracranial space occupying blood or swelling is the trauma craniotomy, in which a large question mark fronto-temporo-parietal myocutaneous flap is turned in order to lift a craniotomy of minimum dimensions 12−15 -15 cm,

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providing access to a large component of the ipsilateral supratentorial compartment and allow for a large volume decompression as appropriate (Figure 3).

With space-occupying intracranial blood (EDH, ASDH, ICH) the goal of surgery is to evacuate the haematoma and to restore ICP homeostasis. Isolated EDH is less commonly associated with significant brain swelling, and generally the bone flap can be replaced following clot evacuation. EDH can expand in volume rapidly; indications for EDH evacuation are haematomas of volume >30 ml regardless of GCS, and if the patient is in coma or has pupil inequality then surgery should occur without delay. An EDH can be carefully monitored non-operatively in patients with GCS >8 if the haematoma volume is <30 ml, maximal haematoma thickness <15 mm and MLS <5 mm.

In contrast, ASDH is often associated with a swollen brain, and a decompressive craniectomy (DC) is not infrequently performed to account for expected postoperative brain swelling. In a similar fashion to EDH, indications for SDH evacuation include haematoma thickness >10 mm and/or MLS >5 mm regardless of GCS, and in patients in coma who have a decrease in GCS of 2 or more points between time of injury and hospitalization with CT measurements that do not otherwise meet aforementioned surgical criteria. Like EDH, surgery for SDH should be performed as soon as possible.

Whether or not replacing the bone following evacuation of the ASDH is currently the object of discussion following the publication of a randomized clinical trial comparing the role of decompressive craniectomy vs craniotomy for ASDH. To date, both surgeries have a similar rate of complications and similar outcomes, consequently decompressive craniectomy should be avoided, when possible.[20]

Surgery for ICH should be carried out when the mass effect related to haematoma (and/or its surrounding oedema) leads to progressive neurological deterioration and to refractory intracranial hypertension. Both haematoma evacuation or DC are surgical options and they may be performed concurrently. CT-derived measurements that warrant surgery include frontal or temporal ICH with volume >20 ml and midline shift of >5 mm or

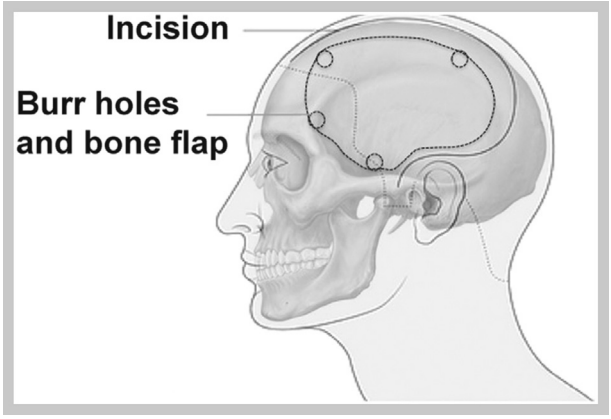

Figure 3 The trauma craniotomy. Unilateral pathology can be decompressed with a large area craniotomy extending from the keyhole anteriorly, the squamous temporal bone inferiorly and the parietal bone posteriorly. The medial border should be lateral to the superior sagittal sinus. Bilateral pathology may be better served by a bifrontal decompression.

basal cistern compression in patients who are GCS 6–8, or ICH volume >50 ml irrespective of other clinical criteria.

The management of skull fractures, involving both skull base and skull vault, can be often conservative. Linear and minimally displaced fractures tend to heal spontaneously and surgery is not required. Indications for surgery are represented by concerns for increased risk of infections (e.g. open wound overlying the fracture edges) or if the fracture fragments are responsible for neurological deterioration. Depressed but closed fractures represent a grey area as they can be managed non-operatively in the absence of neurological deficit secondary to CNS irritation. Open fractures with depression greater than the cranial thickness should be elevated and repaired to minimize the risk of CNS infection. Open fractures should only be managed non-operatively should they show no clinical or radiological evidence of dural breach, have no associated significant intracranial blood load, the depression is less than 10 mm, the frontal air sinuses are not involved, there is no gross cosmetic deformity or pneumocephalus, and there is no wound infection or gross contamination.

Penetrating brain injuries are often treated as with open depressed skull fractures, with the additional factors of ICH, contaminated foreign bodies that need debriding, and the possibility of exit wounds, such as in missile injuries.

If surgical indications are not met initially, many TBI patients who are ventilated and/or are unable to be monitored neurologically will require insertion of an ICP monitor as a surrogate for clinical evaluation. Continuous ICP monitoring allows for targeted treatment within the critical care setting of both ICP and CPP. Some centres will also have access to multimodality monitoring, in which case therapy can also be targeted towards other physiological markers, such as brain tissue oxygenation and microdialysis. Indications for ICP monitoring include salvageable patients with GCS 3–8 and an abnormal CT scan, or a normal scan and two or more of the following: age >40 years, unilateral or bilateral motor posturing, or systolic blood pressure <90 mmHg. An ICP monitor is also frequently inserted in conjunction with any of the above operations as a perioperative adjunct, rather than as a standalone procedure. Delayed surgery can result from ICP targeted therapy, including insertion of external ventricular CSF drainage devices (EVD), or DC. DC is effective at treating medically refractory intracranial hypertension and decreasing severe TBI mortality but with a spectrum of outcome categories, and the decision to proceed to DC can be controversial (see below).

Neurocritical care and perioperative management

Acute surgical interventions are performed to evacuate intracranial mass lesions and restore ICP homeostasis; and neurocritical care, on the other hand, provides supportive measures to minimize secondary brain injury by manipulation of physiological parameters assisted with targeted therapy until the period of deranged intracranial dynamics has resolved.

In most modern neurocritical care units (NCCU), the fundamental pathophysiological parameters that therapy is targeted towards are ICP and CPP. Although much of the evidence has been observational, intracranial hypertension is associated with poorer prognosis in TBI and routine monitoring in individuals who meet criteria for monitor insertion is recommended to reduce TBI mortality.[21,22] Thresholds for ICP intervention vary

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between clinical units; however, most accept an upper range of 20–25 mmHg, with the official upper threshold currently recommended in the BTF guidelines being 22 mmHg.[21] It is also important to remember that these are mean pressure figures, and because ICP can be so dynamic it may take 30 minutes of data to obtain accurate mean values. What is more, there is significantly more data that can be ascertained from ICP waves than just mean pressures, including wave patterns, pulse amplitude and pressure reactivity index, and at present ICP directed guidelines take none of this extra data into account.[23]

The other major physiological parameter targeted, and for which knowledge of ICP is critical to calculate, is CPP. As already demonstrated, CPP is the difference between mean arterial blood pressure and ICP and is tightly autoregulated across a range of physiological blood pressures in the non-injured brain (Figure 1b). In the acute TBI brain, however, autoregulation of blood flow can be severely compromised, and CPP targeted therapy is aimed at maintaining cerebral oxygen and nutrient supply. The original high (>70 mmHg) CPP targeted therapies were associated with an increased risk of respiratory complications and poorer outcomes, hence guidelines for optimal CPP are now in the range of 60–70 mmHg, and aggressive targeting of CPP >70 mmHg is no longer recommended.[21] What is being more and more appreciated, however, is that ICP and consequently CPP are much more than just numbers, and in future therapy may be more specifically targeted to optimal physiological parameters for an individual, rather than a pre-determined threshold or range.[23] Multimodality monitoring, including brain tissue oxygenation (PbtO2) and microdialysis, may have a role in improving individualized targeted therapy. The use of multimodality monitoring, in particular PbtO2, remains controversial and the subject of ongoing investigation; Phase II randomized clinical trials[24] have suggested that monitoring PbtO2 together with ICP leads to improved patient morbidity and mortality following TBI which has led to several Phase III studies,[25,26] whereas other randomized controlled trials have failed to demonstrate any improvement in neurological outcomes with the addition of PbtO2 monitoring.[25] It is also important to note that multimodal monitoring is still not accessible in many developing countries due to its associated high cost and the intensive care facilities required to run them.

Endotracheal intubation and mechanical ventilation are important in the comatose TBI patient, in whom airway protection and respiratory drive are frequently compromised. Furthermore, tight control of ventilation allows for manipulation of partial pressures of arterial oxygen and carbon dioxide (CO2). CO2 is a potent vasodilator, and when ICP is high and/or cerebral compliance low, hyperventilation may be beneficial to vasoconstrict cerebral vessels and therefore reduce intracranial blood volume.[5] The cost of this, however, is of arteriolar vasoconstriction and potentially cerebral hypoxia, hence hyperventilation is now utilized predominantly as an ICP temporizing measure, with prolonged hyperventilation to arterial partial pressure of CO2 to _25 mmHg not recommended.[21]

Hypoxia is to be avoided, for both prevention of secondary hypoxic injury and also hypoxic cerebral vasodilation.[5] Patients who are slow to extubate may require insertion of a percutaneous tracheostomy to promote weaning from mechanical ventilation, aid secretion clearance and prevent complications from prolonged endotracheal intubation, although tracheostomy does not reduce mortality or nosocomial pneumonia.[21]

Sedative drugs have multiple benefits in the TBI patient. The primary benefit is of cerebral metabolic suppression, which can reduce oxygen demands of the injured brain, that subsequently reduces cerebral blood flow, cerebral blood volume and ultimately ICP. Although severe TBI patients are by definition in coma they may still be intolerant of an endotracheal airway, and sedation decreases this irritability, promotes mechanical ventilation and suppresses the cough and gag reflex, all of which can help reduce ICP.[5] Intravenous sedation with propofol or midazolam are common first-line sedative agents, often supplemented by an opioid analgesia such as fentanyl or remifentanyl; however, each drug has its advantages and disadvantages, particularly with prolonged use.[5] Of note, propofol can result in hypotension which must be strictly avoided, with current blood pressure recommendations being maintenance of systolic blood pressure >100 mmHg for patients aged 50–69 years or >110 mmHg for those aged 15–49 years or over 70 years to decrease mortality and improve outcome.[21] Another useful feature of shorter acting intravenous sedative, hypnotic and analgesic agents is that they allow for temporary sedation hold in order to reassess neurology and potential emergence from TBI coma in order to progress to the next stages of management.

Somewhat against the Monro–Kellie doctrine of a constant volume brain, cerebral fluid load can be manipulated by the use of hyperosmotic therapy. The two commonly used hyperosmolar agents used are mannitol and hypertonic saline (HTS). Both will reduce cerebral fluid volume by osmosis into the vasculature; however, both probably also contribute to lowering ICP secondary to reduced blood viscosity, improved rheology and microcirculation and overall reduced cerebral blood volume. Mannitol is typically administered at 0.25 g/kg to 1 g/kg body weight, and hypertonic saline administered as intravenous boluses of concentrations up to 20%. Whereas previously neither agent was recommended over the other, current guidelines have suggested that HTS is preferred for the treatment of cerebral oedema in TBI although the quality of evidence is low.[26]

Temperature regulation has multiple potential beneficial effects. Cooling to hypothermia has shown to be neuroprotective in animal studies, yet these effects are yet to translate into clinical trials, and prophylactic hypothermia is not recommended.[21] Cooling can certainly be effective in reducing ICP and is a successful tool in ICP directed therapy. Finally, fever has been associated with poorer outcomes, and preventing fever by cooling may have a beneficial role.[5]

Although hyperglycaemia may be associated with poorer outcomes in TBI, tight glycaemic control may be associated with periods of low brain glucose which may in fact be more detrimental. As such, the focus should be on hyperglycaemia avoidance rather than tight glycaemic control.[5] Energy expenditure is increased following TBI, and feeding to attain basal caloric replacement should commence by at least day 5 post-injury.[21]

PTS are also a common event postinjury; they may be distinguished in early PTS (within the first 7 days from trauma) and late PTS (after 7 days from trauma). Some studies also distinguish immediate PTS (within the first 24 h from trauma). Overall, clinical seizures can affect up to 16.9% and subclinical seizures up to 25%.[21] PTS may have a deleterious effect on the

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injured brain and may be associated with intracranial hypertension, although they have not been shown definitively to contribute to poorer outcomes.[21] Despite that, PTS seem to impact length of stay and consequently they increase the cost of management of TBI. At present there is a very poor level of evidence regarding the use of antiepileptic prophylaxis in TBI, with no apparent advantage of newer antiepileptics such as levetiracetam over older agents such as phenytoin, despite a more acceptable adverse drug profile.[21,27] There is also an even bigger literature gap regarding the prevention and management of late post-traumatic epilepsy, so it is currently impossible to determine guidelines for these clinical events.[5,13] To date, BTF guidelines are still recommending phenytoin in the first 7 days from TBI, only at discretion of the clinicians involved. The poor level of evidence and the large of number of biases and confounding factors affecting studies on PTS makes it almost impossible to identify potential risk factors and consequently develop a predictive model.

Deep venous thrombosis (DVT) is very common following TBI and is preventable with the use of low-molecular-weight heparin or low-dose unfractionated heparin, but at the cost of an increased risk of intracranial haematoma expansion.[21] The timing of commencement of chemical DVT prophylaxis needs to be made with consideration of the risk of haematoma expansion, and alternative prophylactic therapies such as mechanical stockings or inferior vena cava filters may be considered.

In TBI patients with intracranial hypertension that has not responded to surgical intervention or simple measures, ICP targeted therapy can progress in a stepwise fashion (Figure 4). Many interventions to reduce ICP are associated with adverse effects, and these must be balanced against poor outcome with persistent intracranial hypertension.[5] With each advance in ICP driven protocol the patient must be reassessed by an experienced clinician and reimaged to look for evacuable space occupying lesions or CSF diversion. As the acute intracranial pathology settles, neurocritical intervention can be de-escalated with a stepwise and controlled withdrawal of paralysis, cooling and sedation. Careful monitoring of ICP, CPP and clinical parameters such as motor response and pupil diameter and reaction to light is important during de-escalation to ensure that it is not being performed prematurely. Continual reassessment of the patient, their examination findings, blood results, physiological parameters and radiology findings are paramount to detect changes in cerebral health and manipulate physiological parameters towards recovery. Once patients are transferred to a general care ward it is important to engage in rehabilitation services early, rehabilitation needs and goals are identified, and transfer to a dedicated neurological rehabilitation centre arranged as appropriate.

Conversely, in those that do not respond to appropriate stepwise measures, or who have clinical, physiological or radiologic findings incompatible with meaningful recovery, end-of-life discussions with family are appropriate and comfort measures initiated in line with local customs and policies.

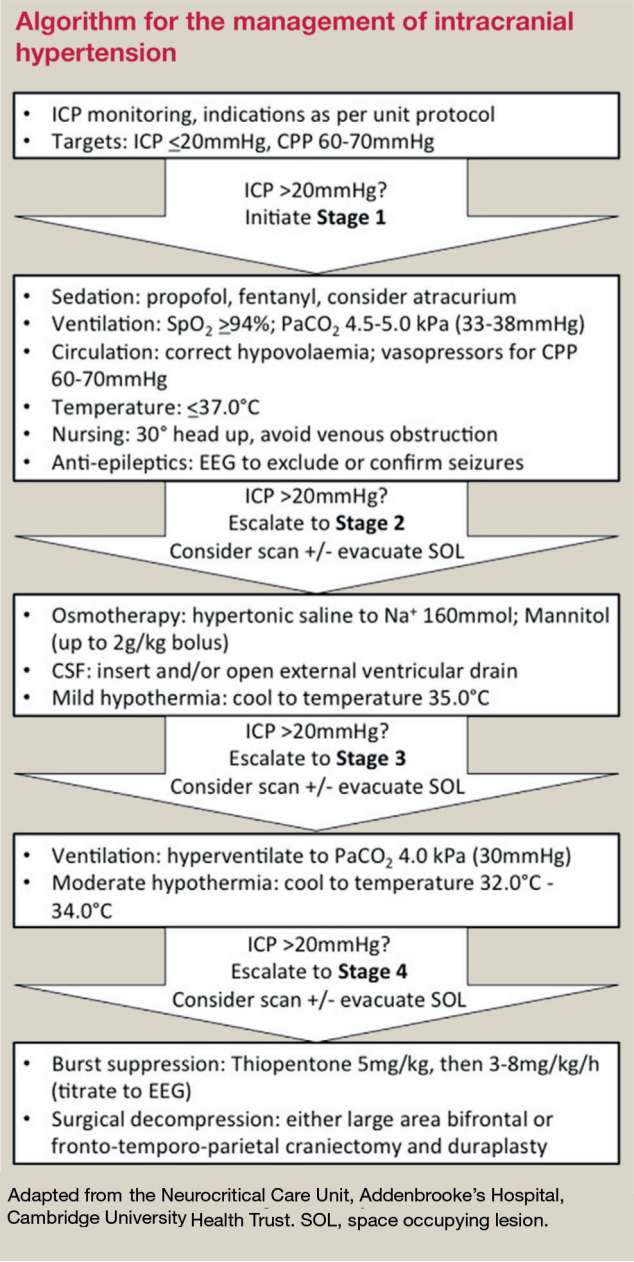
Algorithm for the management of intracranial hypertension
ICP monitoring, indications as per unit protocol
Targets: ICP ≤20mmHg, CPP 60-70mmHg
ICP >20mmHg?
Initiate Stage 1
Sedation: propofol, fentanyl, consider atracurium
Ventilation: SpO2 ≥94%; PaCO2 4.5-5.0 kPa (33-38mmHg)
Circulation: correct hypovolaemia; vasopressors for CPP 60-70mmHg
Temperature: ≤37.0°C
Nursing: 30° head up, avoid venous obstruction
Anti-epileptics: EEG to exclude or confirm seizures
ICP >20mmHg?
Escalate to Stage 2
Consider scan +/- evacuate SOL
Osmotherapy: hypertonic saline to Na+ 160mmol; Mannitol (up to 2g/kg bolus)
CSF: insert and/or open external ventricular drain
Mild hypothermia: cool to temperature 35.0°C
ICP >20mmHg?
Escalate to Stage 3
Consider scan +/- evacuate SOL
Ventilation: hyperventilate to PaCO2 4.0 kPa (30mmHg)
Moderate hypothermia: cool to temperature 32.0°C - 34.0°C
ICP >20mmHg?
Escalate to Stage 4
Consider scan +/- evacuate SOL
Burst suppression: Thiopentone 5mg/kg, then 3-8mg/kg/h (titrate to EEG)
Surgical decompression: either large area bifrontal or fronto-temporo-parietal craniectomy and duraplasty
Adapted from the Neurocritical Care Unit, Addenbrooke's Hospital, Cambridge University Health Trust. SOL, space occupying lesion.

Figure 4

Decompressive craniectomy for refractory intracranial hypertension

Despite the publication of two well-designed ICP-based multicentre randomized controlled clinical trials, the role of DC in TBI remains a controversial and frequently emotive topic. In the Australasian DECRA trial,[28] salvageable adult patients with TBI, GCS 3–8 and ICP above 20 mmHg for more than 15 minutes refractory to first-tier therapies were randomized to either continued medical management or DC. The intervention group showed less time with ICP above the threshold, required fewer interventions for raised ICP and had fewer days in intensive care, but ultimately they also had worse 6 month Glasgow Outcome Score and also a higher risk of unfavourable outcome, with no increase in mortality. Differences between baseline characteristics, such as more severe injuries amongst the surgical cohort, and high crossover rates made a general consensus statement regarding the role of DC following TBI and ICP >20 mmHg difficult to currently establish.

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The European RESCUEicp trial was larger, had a higher ICP threshold (25 mmHg), and refractory intracranial hypertension was tolerated longer (from 1 to 12 hours).[29] The authors demonstrated that DC for persistently raised ICP was associated with lower 6 month mortality than with medical management alone, but higher rates of vegetative state and severe disability.

Data from both of these trials suggest that DC, despite being a life-saving operation, is not necessarily associated with acceptable functional survival. With the understanding that the RESCUEicp results would be available following the release of the current BTF guidelines, an update was published in 2020 to incorporate the new data.[30] As such, the new recommendations (level IIA evidence) now include the following: secondary DC performed for late refractory ICP elevation is recommended to improve mortality and favourable outcomes; secondary DC performed for early refractory ICP elevation is not recommended to improve mortality and favourable outcomes; and secondary DC, performed as a treatment for either early or late refractory ICP elevation, is suggested to reduce ICP and duration of intensive care, though the relationship between these effects and favourable outcome is uncertain. How these updated guidelines ultimately impact clinical practice is yet to be determined.

The role of craniotomy with haematoma evacuation compared with additional DC in the management of ASDH was the main focus of an international randomized clinical trial (RESCUE-ASDH). This study demonstrated that patients with DC had no better clinical outcomes compared with patients who underwent craniotomy for haematoma evacuation and reinsertion of the bone flap. The results of this study have not yet been incorporated in the latest version of the BTF guidelines, however they open the debate over possible alternatives to decompressive craniectomy, such as hinge craniotomy.

Terminal care

Despite best medical practice, severe TBI is not infrequently a terminal event. Futile situations can be easier to recognize than situations in which survival is possible but clinical outcome is uncertain. When brain injury is widespread or when a favourable outcome is doubtful and enough time has elapsed for an experienced clinician to prognosticate, then initiation of palliative measures may be appropriate in line with the patient's known or expected wishes and after extensive family discussions. It is extremely important to remember that what different individuals will accept in terms of disability is highly variable, as is the definition of 'favourable' clinical outcome, and this is a source of ongoing debate in the literature.[31] Clinical and/or radiological brain death testing will often help aid the decision process. Tools to estimate clinical outcome are available and will no doubt be augmented in the future with advances in artificial intelligence. Depending on local laws and customs organ procurement may be an option for brain-dead patients, which can be a sensitive topic of discussion and often best left for specialist organ donation clinicians. Families of severe TBI victims should be offered grief counselling and support.

Long-term management

A broad range of TBI survivors may benefit from dedicated neurorehabilitation following step down of their medical needs.

Cognitive and behavioural deficits may be just as debilitating as physical impairment. Recognition of higher function deficits can be an important step in directing patients towards neuropsychological screening assessment and rehabilitation. Return to employment or study may be challenging, however supported return to previous level of function may also aid in overall rehabilitation and feelings of self-worth. Chronic and sometimes debilitating headaches are not infrequently reported which do not always respond to conventional methods, and reduced stamina with increased fatigability are also common findings.

Neuroendocrine disturbance is not particularly common, but pituitary function should be screened for to exclude an organic cause for symptoms.

There is increasing evidence to suggest that TBI is a chronic and evolving condition rather than an isolated event. The overall late mortality of TBI remains unknown and it represents a challenge for future research. TBI also seems to be a risk factor for later onset neurodegenerative disease, with increasing interest in the relationship between mild injury such as sports-related concussion and the development of Alzheimer's disease.[32]

Finally, reconstructive cranioplasty for those who have undergone decompressive craniectomy is recommended to provide protection for the underlying brain, improve cosmetic appearance, aid patient psychology and reintegration within the community, and also potentially has a role in neurologic improvement by improving abnormal intracranial physiology associated with the syndrome of the trephined. The ideal material and timing for cranial reconstruction remain uncertain despite the large number of papers published in the last few years. Further studies are required to determine the efficacy and safety of early cranioplasty.[33]

Advances and future directions

Our knowledge on the management of TBI has been greatly advanced by randomized clinical trials (RCT), and several current trials are worth mentioning. Post-traumatic seizures are presently being investigated by the various MAST (Management of Seizures post-Traumatic brain injury) trials, incorporating two parallel RCTs aiming to optimize clinical practice of anti-epileptic agents following TBI. The original studies that demonstrated seven days of anticonvulsant use following TBI was associated with a reduction in early PTS used phenytoin as the trial drug whereas many clinicians currently use levetiracetam as their anticonvulsant agent of choice owing to its vastly superior adverse effect profile, yet levetiracetam remains unproven in this setting. The MAST trials aim to investigate both phenytoin and levetiracetam for this purpose, as well as the role of these drugs in the prevalence of delayed PTS up to two years following TBI.[34]

There are currently two parallel RCTs investigating the role of PbtO2 monitoring following severe TBI, the Australian and New Zealand based global study (BONANZA-GT), and a North American study (BOOST-III). In a blinded fashion, patients in both studies will have clinical outcomes compared in two separate arms, with one group of patients receiving standard ICP-driven care, and the other group receiving standard care plus additional treatment aimed to prevent cerebral hypoxia as monitored by an intracranial PbtO2 monitor. Although seemingly intuitive and already adapted in some neurocritical centres,

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treatment aimed at limiting cerebral hypoxia as measured by a PbtO2 monitor are not yet proven and these important studies will hopefully provide insights into their role in the improvement of clinical outcomes following severe TBI.[35,36]

Medicine in general is currently undergoing rapid advancements and for the foreseeable future much of this will be driven by machine learning (ML) and artificial intelligence (AI). The management of TBI is no exception to this and treatment guidelines will no doubt incorporate algorithms developed by the machine learnt analysis of vast volumes of radiological and physiological data relating to TBI. Identification of patients at risk of secondary brain injury can be challenging, and the optimal outcome may be achieved by application of an individual treatment strategy developed by AI incorporating initial scan results and measured pathophysiological parameters. AI models also could incorporate new data and modify treatment strategies extemporaneously with rapid continuous adjustments, and as monitoring equipment becomes more sophisticated so too will the management algorithms. This could involve minor changes such as a small modification in a ventilator setting, or larger interventions such as the decision if or when to perform a DC in certain clinical settings. Data acquisition will be key to refining these models, and observing their application in randomized controlled trials will demonstrate their ultimate worth.

Conclusions

TBI remains a huge global socioeconomic issue. Protocols involving resuscitation, management strategies involving neurocritical care, and radiological diagnosis are all useful resources aiming to improve clinical outcomes by minimizing secondary brain injury. Management is not limited to the traumatic event, but it may last for months or years, with involvement of physical, cognitive and neurobehavioral rehabilitation. A progressively increasing knowledge of TBI pathophysiology is shifting the paradigm of treatment from damage control to reparative treatment, but this process remains a great challenge for the future. Major clinical trials are currently recruiting and the results may significantly contribute to this purpose. The use of AI is still in its early stages but it may be of great help to improve the management of TBI. ◆

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Practice points

- Patients with TBI should be assessed and managed as early as possible to avoid secondary brain injury. Early classification according to GCS should be used due to its predictive value. Pupillary response should be also monitored
- CT is still the gold standard for radiological recognition of TBI and it should be performed as soon as possible to expedite and target patient management
- Surgery could be performed to evacuate a space-occupying lesion (EDH, ASDH, contusion/ICH) and/or decompress a swollen brain (decompressive craniectomy). In this scenario, a trauma flap (12 x 15 x 15 cm) should be performed. Fractures can often be managed conservatively except when there is a concern for infection (open fractures) and/or the fracture is responsible for neurologic deterioration
- Neuromonitoring should be inserted in severe TBI and/or TBI patients who are ventilated and/or are unable to be monitored neurologically
- Neurocritical management mostly targets ICP (<20 mmHg) and CPP (between 60 and 70 mmHg). Hypotension, hypoxia, hypercapnia, hyperthermia and fluid overload should also be minimized as part of intensive care management
- There is a low level of evidence supporting the use of prophylactic antiepileptic drugs in TBI
- Neuropsychological assessment and rehabilitation should always be considered after TBI

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